

Many trace elements are investigated for their effect on LPL activity. Some increase the enzyme activity (e.g., Al, Cr) whereas some others, such as Mn and Li, lead to decreased activity of the enzyme. A major function of the liver is its ability to act as a buffering system for many biomolecules including lipids. Trace elements are also shown to change the content of triglycerides and phospholipids through changes in the rate of synthesis and/or release. There are reports that some elements protect the cells from the toxicity of others. For example, Cu is shown to reduce the Al-induced toxicity on lipid metabolism.

Summary #107

ANTIOXIDANT ACTIVITY OF PLASMA BILIRUBIN PROTECTS AGAINST DRUG-INDUCED OXIDATIVE STRESS IN GROWING RAT LIVER

Abdolamir Allameh, Faezeh Fatemi, Abolfazl Dadkhah, Mohammad Rahmati, Department of Clinical Biochemistry, Faculty of Medical Sciences, Tarbiat Modarres University, POB 14115-331, Tehran, Iran. E-mail: allameha@modares.ac.ir.

Our goal was to investigate the role of bilirubin on total antioxidant activity (TAC) of plasma and modulation of liver damages in weanling and adult rats. Recently we reported that TAC of plasma, determined as ferric reducing ability of plasma (FRAP), is remarkably induced in developing rats pretreated with high dose vitamin K1 or menadione. Increased FRAP was inversely related to formation of lipid peroxidation products in plasma and erythrocytes. In the present study, the role of selected antioxidant factors in the drug-related changes in FRAP was examined. FRAP was monitored in control and rats given different doses of acetaminophen (0, 25, 250 or 450 mg/kg BW). In parallel, the levels of enzymatic and non-enzymatic antioxidant parameters were measured. The antioxidant activity was evaluated in relation to the rate of lipid peroxidation products in liver. Unlike adults, in growing rats treated with a sublethal dose of acetaminophen (450 mg/kg bw) there was about a 4- to 5-fold increase in FRAP. Under these circumstances, no significant changes were recorded in total protein and uric acid levels in plasma as well as catalase activity in erythrocytes. In contrast, an increased FRAP value was associated with plasma bilirubin as well as erythrocyte superoxide dismutase (SOD) activities. Kinetic studies showed that FRAP was elevated during 1–4 h after acetaminophen treatment. Increased FRAP occurred simultaneously with a surge in total bilirubin 6 h after acetaminophen administration. However, elevation in SOD in erythrocytes occurred with a delay (12 h after drug administration). These results suggest that in immature rats, plasma bilirubin and erythrocyte SOD play a distinct role in elevation of total antioxidant capacity of plasma leading to suppression in the level of lipid peroxidation products in plasma.

Summary #108

RELATIONSHIP BETWEEN HYPERHOMOCYSTEINEMIA, OXIDATIVE STRESS AND SEVERITY OF ATHEROSCLEROTIC LESIONS

Refahi Rogyeh, Refahi Soheila, Mardi Afrouz, Mashoufi Mehrnaz, Jabbarzadeh Tahereh Quality Control Laboratories, Emam Khomeini St., Tehran, Iran. E-mail: refahi84@yahoo.com.

The aim of this study was to evaluate the correlation between hyperhomocysteinemia and oxidative stress with the severity of atherosclerotic lesions. The patients (52 males and 36 females) were selected from individuals with angiographically defined CAD admitted to the Shahid Madani Hospital. Control groups were selected from sex- and age-matched apparently healthy individuals (15 males and 24 females). Serum homocysteine levels were measured by an ELISA method (Awareness stat fax-2100 Model). Total antioxidant capacity of samples was determined by an autoanalyzer (COBAS MIRA plus model) using Randox kits. The correlation between the measured parameters and extension of atherosclerotic lesions were calculated using statistical analysis in both groups. The mean levels of homocysteine in patients and the control group was $20.38 \pm 10.22 \mu\text{mol/L}$ and

$18.56 \pm 9.47 \mu\text{mol/L}$, respectively, and the differences were not significant ($P > 0.05$). In the patient group, the mean level of antioxidant capacity was $1.34 \pm 0.12 \text{ mmol/L}$, whereas that of the control was $1.39 \pm 0.12 \text{ mmol/L}$ ($P > 0.05$). Using statistical analysis, no correlation was noticed between the extension of atherosclerotic lesions, total antioxidant capacity and serum total homocysteine levels. In this study no significant differences between levels of homocysteine and total antioxidant in patients and the control group were found, along with no marked relationship between the studied parameters, which suggests that hyperhomocysteinemia has no important role in the progression of atherosclerotic lesions.

Summary #109

LIPIDS, LIPOPROTEINS, APOLIPOPROTEINS, AND LIPOPROTEIN PROFILE IN PATIENTS WITH CORONARY HEART DISEASE REFERRED TO THE YAZD CARDIOVASCULAR RESEARCH CENTER

B. A. Jalali-Khanabadi, H. Mozaffari-Khosravi, M. Rafiei, M. Nemayandeh, F. Darabi, Biochemistry Department, Faculty of Medicine, Yazd University of Medical Sciences, Yazd, Iran. E-mail: bajalali@yahoo.com.

Coronary heart disease (CHD) is the leading cause of death in many populations, including Iranians. The best way to control CHD is identify and modify more effective local risk factors. The aim of this study was to determine and compare lipids, apolipoproteins, and lipoprotein(a) [Lp(a)] in patients with CHD referred to the Yazd Heart Research Center. One hundred patients (37 females, 63 males) with CHD, and 92 healthy controls (58 females, 34 males) were investigated. The fasting plasma total cholesterol (TC), triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C), were determined immediately by routine laboratory methods. Lp(a), apo-A1 and apo-B100 were determined after collection of all blood samples by electroimmunoassay. The statistical tests included a *t* test for comparison of lipids, lipoproteins and apolipoproteins and a *U* test for comparison of Lp(a) in two groups. TC and low-density lipoprotein cholesterol in patients (227.3 ± 44.7 and $147 \pm 29.7 \text{ mg/dL}$, respectively) were higher than controls (207.5 ± 54 and $127 \pm 39 \text{ mg/dL}$). There were not any significant differences in TG, HDL-C and apoA1 between the two groups, but apoB100 was higher in patients ($1.25 \pm 0.41 \text{ g/L}$) than controls ($1.14 \pm 0.36 \text{ g/L}$). Lp(a) was higher in patients ($25.1 \pm 27 \text{ mg/dL}$) than controls (18.86 ± 19.6) but was not statistically significant ($P = 0.067$). We concluded that high levels of cholesterol and cholesterol-rich lipoproteins are more associated with CHD. These lipids and lipoproteins may be more effective local risk factors for this disease in Iranian populations.

Summary #110

PURIFICATION OF PROTEIN CONTAINS A SIX HISTIDINE TAG USING A METAL CHELATING RESIN, NI-SP50

A. Mohsenifar,¹ A. S. Lotfi,¹ B. Ranjbar,² A. Allameh,¹ S. Hasannia,³ F. Rahbarizadeh,¹ K. Omidfar,¹ M. Nikkha,⁴ A. Khoshdel,¹ B. Etemadi Kia,¹ ¹Department of Clinical Biochemistry, Faculty of Medical Sciences, Tarbiat Modares University; ²Department of Biophysics, Faculty of Basic Sciences, Tarbiat Modares University; ³Department of Biology, Guilan University, Rasht; ⁴Department of Biochemistry, Biophysics and Biochemistry Institute, Tehran University, I.R. Iran

A six histidine tag allows the immobilization of the protein on metal chelating surfaces. This approach facilitates purification of expressed protein which contains a six histidine tag in the N-terminal or C-terminal region of the protein just in one step. Our aim was to identify the possibility of using the cation exchanger resin SP50 for purification of protein which contains a six histidine tag. *E. coli*, BL21 and M15 strains were transformed with the plasmid, PQE31, containing the cDNA for human neuroserpin. After induction by IPTG (2 and 1 mM) in 2XYT medium for four hours, the cells were harvested by centrifugation and were disrupted by sonication. Inclusion bodies were washed three times by inclusion body washing buffer (Tris base 50 mM, Triton X-100 0.5%, pH 7.8) prior to solubilization in

CLINICAL BIOCHEMISTRY

Volume 38
Number 9
September 2005

Review

Shane Miersch and Bulent Mutus

777 Protein S-nitrosation: Biochemistry and characterization of protein thiol-NO interactions as cellular signals

Clinical

Hafize Uzun, Melek Ozmen Keles, Rezzan Ataman, Seval Aydin, Betül Kalender, Ezel Uslu, Gönül Simsek, Metin Halac, and Safiye Kaya

792 Serum cystatin C level as a potentially good marker for impaired kidney function

Edward W. Randell, Maria S. Mathews, Hongwei Zhang, Jim S. Seraj, and Guang Sun

799 Relationship between serum butyrylcholinesterase and the metabolic syndrome

Dick C. Chan, Gerald F. Watts, Theodore W.K. Ng, Yoshiaki Uchida, Naohiko Sakai, Shizuya Yamashita, and P. Hugh R. Barrett

806 Apolipoprotein B-100 kinetics and static plasma indices of triglyceride-rich lipoprotein metabolism in overweight men

Ionela Gheorghiu, Andriy Moshyk, Raymond Lepage, Charaf E. Ahnadi, and Andrew M. Grant

813 When is bioavailable testosterone a redundant test in the diagnosis of hypogonadism in men?

Monica Campagnoli, Alberto Sala, Sara Labò, Antonio Rossi, Thomas J. Neuhaus, Christian P. Braegger, Lorenzo Minchiotti, and Monica Galliano

819 Analbuminemia in a Swiss family is caused by a C → T transition at nucleotide 4446 of the albumin gene

continued on back cover



ELSEVIER

ISSN 0009-9120

INDEXED/ABSTRACTED IN: Current Contents/Life Sciences, Index Medicus, MEDLINE, MEDLARS, BIOSIS Database, Chem Abstracts, Current Awareness in Biological Sciences (CABS), Reference Update